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Influence of Nutrition on Parodontal Health

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During recent years an increasing number of investigations has been made of the state of nutrition of communities in different parts of the world. These field surveys were often instituted as a result of the recognition of certain fairly well defined disease syndromes occurring in regions where, for climatic, geographic, economic or other reasons, gross dietary deficiencies obtained. At the same time, laboratory studies were directed to the production, prevention and cure of apparently analogous lesions in experimental animals. In many instances the laboratory workers took as their guide some peculiarity in the diet of the people suffering from the particular lesions under investigation. The great advantage of the experimental approach lay in the controlled nature of the conditions which could be imposed, and this method of study has been amply justified by the knowledge so gained of the causes of beriberi, scurvy, rickets, pellagra and other diseases of nutritional origin. Later, it became recognized that the absence of *obvious* clinical signs of deficiency diseases in any given community did not necessarily imply that the nutrition of that community could be considered adequate for the maintenance of positive health. Laboratory experience pointed to the value of certain specific dietary factors in stimulating the natural resistance of the organism to various pathological lesions, including some bacterial infections, which in themselves could not be labelled true deficiency diseases. It was further noted that diets containing quantities of some food factor sufficient to prevent gross disease might still be inadequate for perfect tissue structure, health and function.

As a clinical sequence to this changing conception of nutritional science, the so called nutrition survey has expanded into a much more elaborate and often unwieldy undertaking. In addition to assessment of the dietary of representative families or individuals and to recording of gross defects in health, exhaustive physical examinations are now made, urine samples are studied, and specimens of blood are taken for haemoglobin estimation, cell counts, and measurement of content of minerals, vitamins and other substances; even the maternal milk has not escaped detailed study. The clinical spotlight has become focussed on

the search for evidences of sub-optimal health rather than established disease. Much therefore depends upon the development of diagnostic methods of a high degree of accuracy. Radiography, biopsy examination of the skin especially in relation to the hair follicles and sweat glands, studies of dark adaptation of the eye, observation of the cornea and tongue with the slit lamp microscope, all have lately received particular attention. Strangely, perhaps, the teeth and their related structures have been somewhat neglected; even when their condition has been noted, the examination criteria have too frequently been so poor as to be useless for scientific purposes, and this is especially true in the case of parodontal disease, with which this communication is largely concerned.

It is as well to mention that the term "parodontal disease" is here used to cover all departures from positive health of the tissues which surround and support the teeth, from slight reddening of the gum margin to gingival atrophy, bone rarefaction and loosening of the teeth. Our knowledge of the connexion between nutritional defects and parodontal disease has been gained almost solely from controlled investigations on laboratory animals and in this field we owe much to the pioneer work of Lady Mellanby. It is true that the suggested application of these experimental findings to the human subject has aroused a good deal of criticism. Nevertheless there are certain experimental indications which cannot be dismissed by mere expressions of opinion or uncontrolled clinical "impressions." I propose, then, to deal very briefly first with the more important animal findings, next with their possible application to disease in man, and finally with the urgent need for the development of more accurate clinical criteria for determining the *incipient* phases of parodontal disease and for assessing response to treatment.

Deficiency Disease in Experimental Animals

The influence of vitamin A can be well illustrated in the dog. As far as the parodontal tissues are concerned, deficiency of this food factor exerts its most drastic effects on the gum and alveolar bone during early life. As regards the gum, one of the first microscopic evidences of abnormality is hyperplasia of the subgingival epithelium attaching the gum to the tooth surface (Mellanby, M., 1927; Mellanby, M. and King, 1934). The alveolar bone is also increased in amount; growth of the dentine of the permanent tooth roots is arrested and in its place a mass of cementum like tissue is deposited; eruption of the permanent teeth is retarded and, when these teeth eventually erupt, their alignment is irregular (King, 1936). In addition, degeneration of the nerves to the teeth and jaws occurs (Mellanby, M. and King, 1934; King, Lewinsky and Stewart, 1938). The end result of all these changes is hypertrophy and, later, atrophy of the gingivae, secondary infection of the gum by micro-organisms, parodontal abscesses, and loosening of the teeth due to bone resorption, a series of events not unlike those associated with so called pyorrhoea in man. The precise sequence of the changes in experimental vitamin A deficiency is still not clear but there is evidence that the nerve lesions are the result of compressive trauma from abnormal growth of the inner wall

of the bony channels through which the nerves pass (Mellanby, E., 1938, 1941, 1943).

Comparatively few detailed studies have been made of the effects of deficiency of the vitamin B complex on the dental or parodontal tissues. This is surprising in view of the large number of investigations on, for example, the experimental production of blacktongue in dogs, on which much of our knowledge of pellagra is based. My personal experience of canine blacktongue is limited to less than a dozen animals, but the oral observations so far made may add something to those recorded by previous workers. This disease, induced by diets deficient in vitamin B complex, is generally characterized, as far as the mouth is concerned, by reddening of the inner aspect of the cheeks and lips and of the floor of the mouth, pharynx, fauces, tongue and gums; this may progress to irregular, more or less superficial necrosis of these regions. Some of the earlier signs and symptoms include anorexia, loss of weight, injection of the cheek in the neighbourhood of the molar teeth and of the gums, and sometimes a bluish red patch on the dorsum of the tongue near the midline. The colour of the oral mucosa, however, varies from time to time. Sometimes it is bright red and at other times there is a bluish or greyish tinge reminiscent of a subacute or chronic inflammatory process. Just before the onset of the acute phase, a sharp drop in weight is followed by an increased flow of saliva which is ropy and hangs from the corners of the mouth in strings, by injection and haemorrhage of the lip corrugations, and by a foul foetid breath. Then suddenly areas of ulceration appear, often in the cheek, which rapidly involve the whole mouth cavity and are covered with an easily removable, dirty-white membrane. The animal is weak and ill and the ulcerated surfaces are painful to the touch. Administration of nicotinic acid or nicotinamide produces a dramatic improvement and, within about forty-eight hours, little or no sign of the disease can be found and the animal has recovered its appetite and normal activity. Contrary to the opinion of some American workers, I have been unable to associate the most acute phases of the disease with any preponderance of fuso-spirochaetal flora in smears from the ulcerated regions.

Topping and Fraser (1939) studied the effects of deficiency of the vitamin B_2 complex on the oral tissues of the monkey and published illustrations showing severe ulceration of the gums and other tissues of the mouth in animals on diets deficient in nicotinic acid and riboflavin. That the disease was not of infective origin was indicated by its absence when the diet was reinforced by the complete vitamin B_2 complex. Moreover, although the lesions abounded in many types of organisms, inoculation of the necrotic debris into healthy, adequately fed monkeys produced no ill effect, even if the inoculated tissues were previously scarified. It is of interest that these lesions in both dogs and monkeys, caused by deficiency of B_2 vitamins, bear a certain similarity to ulcerative gingivo-stomatitis, Vincent's disease or trench mouth, in man.

Although much work has been done on vitamin C deficiency in guinea-pigs, the fact that this species of animal possesses teeth which grow continuously throughout life has to a large extent precluded its use for studying changes in the gingivae comparable to those in the human being. It is difficult, too, to determine accurately the condition of gum

tissue and alveolar bone through which teeth are continuously passing. As regards other animals, many possess the ability to synthesize vitamin C in their bodies even if the diet be devoid of this food factor. The monkey, however, like the guineapig and man, is also very susceptible to vitamin C deficiency, and Topping and Fraser (1939) report the occurrence of ulcerative lesions of the oral tissues in monkeys given diets deficient in vitamin C.

No mention has yet been made of nutritional factors such as vitamin D, calcium and phosphorus, which affect the structure of bone and other calcified tissues. Obviously, deficiency of these food essentials, by causing rickets, osteomalacia and allied conditions, will produce defective growth of alveolar bone and crowding of the teeth. There are also present in certain cereals substances which still further accentuate faulty calcification, for example, phytic acid, often present in fairly large amounts in oatmeal. On the whole, however, most forms of parodontal disease in animals appear to show their initial lesions in the gum and not the bone and for this and other reasons factors influencing calcification will not be discussed further.

In addition to those already mentioned, there are a number of other vitamins which may play some part in the maintenance of parodontal health, more particularly other components of the vitamin B_2 complex. The published reports concerning them, however, are conflicting. One important point of agreement appears to be that in many animals all of the vitamin B_2 components available in their synthetic form are by no means as effective as the whole complex in the form of less artificial products such as yeast and liver preparations. This, incidentally, is also in agreement with the experience of clinicians in parts of India, Malaya, Africa and elsewhere in the treatment of forms of human stomatitis not usually encountered in this country.

Although perhaps not strictly within the scope of this paper, some mention may be made of the physical character of the diet in relation to gingival health. Many articles have been published in which the authors claim that foodstuffs of a hard and fibrous nature are the controlling factors in parodontal health, irrespective of the chemical composition of the diet. Conversely, soft sticky foods are said to be the chief cause of parodontal disease, because of the retention of food debris about the teeth and of the absence of sufficient gum massage. Real experimental evidence in support of such beliefs is hard to find and, with few or no exceptions, those studies which have so far been made are open to criticism on the grounds of inadequate control. On the other hand, the physical consistency of the food taken into the mouth cannot be lightly dismissed as of no consequence to oral health. A certain amount of roughage is obviously of advantage for promoting normal intestinal movements which aid the absorption of food and the excretion of waste products. There is, too, the possibility that deposition of calculus about the necks of the teeth, which undoubtedly contributes to, or even initiates, certain forms of gingival disease, may be increased or reduced by foodstuffs of different physical consistency. Apart altogether from calculus formation, however, in some instances food particles may cause direct injury to the gum margin, as is well shown in rats fed on coarsely milled oats, the sharp spikes of which penetrate the gum, leading to severe injury and eventual

loss of the teeth (King, 1935). This phenomenon invites speculation as to the damage which may be done to the human gum by undue scouring with hard, dirty toothbrush bristles.

Nutrition and Parodontal Disease in Man

Assessment of the influence of nutrition on parodontal conditions in man presents many unsolved problems. Properly controlled investigations on the human subject are difficult, especially when diet is the chief concern, but until these are undertaken on a fairly large scale little further progress can be made. There is, however, a certain amount of reliable evidence that vitamin C, nicotinamide, and the vitamin B_2 complex as a whole are of no little importance to oral health.

Our information on vitamin A is unreliable. This is hardly surprising, first since frank vitamin A deficiency is uncommon, at least in this country, and second because we know, on the basis of animal experiments, that this food factor particularly affects the development of the tissues and is therefore operative mainly in prenatal life and infancy in respect of the gums and jaws. However, it is justifiable to suggest that a liberal supply of vitamin A for the pregnant and lactating mother and for her child before puberty is an important requirement for the parodontal health of the latter in later life.

With regard to vitamin C, Professor Campbell and Dr. Cook have had more experience than I of the clinical application of the vitamin in diseases of the gums. Personally, I have treated some 80 cases of human gingival disease of varying severity with a fairly wide range of ascorbic acid dosage, with or without supplementary local hygienic measures. Results have been conflicting, if not disappointing, but those of other workers would suggest that the vitamin plays some part in maintaining gingival health and in promoting recovery from disease.

Since 1939 I have obtained some evidence that the nicotinic acid component of the vitamin B_2 complex is of value in the treatment of so called "trench mouth" or Vincent's disease (King, 1940, 1943, 1944, 1). In this condition a fairly widespread ulceration of the tissues of the mouth and throat may occur which in some phases is not unlike that described in animals receiving diets deficient in the B_2 vitamins. In man, a painful haemorrhagic ulceration usually involves the gums and may also affect the pharynx, tonsils, fauces, soft and hard palate, inner aspect of the lips and cheeks, and the tongue. There is often but not always a foul breath and, in the more acute stages, definite malaise, lassitude, anorexia and mental inertia, with enlargement and tenderness of the regional glands. The exact cause of the disease is still unsettled but I believe that the main predisposing agencies are local trauma of the gum, depressed general tissue resistance, temporary vitamin deficiency, *e.g.*, of nicotinic acid, and, coincident with or following these conditions, secondary non-specific bacterial action. The lesions may be treated by attacking any one of these factors separately, but obviously combined operations against all of them are most effective. During the past 5 years I have made detailed records of about 800 cases of trench mouth and have personally treated some 500. Stimulation of tissue resistance does, I believe, play an important part in successful treatment and in

this respect I have found nicotinic acid or nicotinamide of no little value. I have noted that in England the most frequent predisposing factor is the common cold. It is surely no mere coincidence that in this country the number of cases of trench mouth reaches its peak in the autumn and winter, when the common infections, such as colds, are rife, and falls to a minimum in the late spring and summer when tissue resistance is usually highest. There is still a great deal of controversy as to whether or not the lesions of trench mouth are infective. My own opinion inclines to the view that the condition is likely to occur only in persons whose general resistance is depressed by other illness or defective nutrition and whose mouth presents traumatic foci.

The possible relationship of faulty nutrition to other non-ulcerative lesions of the human gum is still far from clear. The common, so called marginal gingivitis is generally believed to be of local traumatic origin, that is, due to irritation of the gum margin by mouth breathing, malocclusion, calculus deposits, food debris, and cervical or interstitial caries, but vigorous local hygienic or surgical measures so frequently fail to eradicate the disease that a systemic basis is implied, and some as yet undetermined nutritional defect may eventually provide the clue.

Diagnostic Criteria

While there is general agreement as to the need for further extensive researches on the basic aetiology of parodontal diseases, little attention has been directed towards improving the methods by which clinical observations of the gingivae can be accurately recorded. In the past too much has depended on the personal experience and opinion of the individual examiner, particularly in regard to differentiation between very early disease and positive health, and assessment of the efficacy of treatment procedures. In other words, we have been seriously handicapped by the absence of any recognized gauge of normality or perfect health. Indeed, the term "normal" is too frequently used almost as a synonym for "the average," and this is true in other branches of medicine. It seems to me more logical to define normality of any tissue or organ of the body as the state of perfection in structure and function at any given stage of development. With this somewhat idealistic definition in mind, for several years I have been trying to determine first those macroscopic and microscopic appearances of the gum which seem to be associated with positive health, and secondly the earlier deviations from health towards disease.

Author's Own Experiments

Recently, I have used the ferret as an experimental animal and, in one form of parodontal disease in ferrets, one of the early clinical changes in the gum proper is a tiny haemorrhage on the buccal carnassial gum. This finding prompted a histological study of the gingival blood supply in health and disease, the method employed being based on the staining of haemoglobin by a mixture of sodium nitroprusside and benzidine followed by oxidation with weak hydrogen peroxide, after Pickworth (1934). I found a close relationship between the vascular condition of the gum

and the state of its epithelial and connective tissues, and a characteristic pattern of the sub-surface capillaries in healthy tissues (King, 1944, 2). The appearances suggested that if the blood vessels could be viewed by some means *in vivo*, a valuable diagnostic aid might be obtained. The slit lamp microscope has for some time been in use for studying the vascularity of the cornea and it was an obvious step to try it for the gum. The usual instrument of this type proved of little use but with the co-operation of Mr. J. Smiles and Dr. E. J. Schuster a more suitable experimental model was constructed. After many trials a technique was developed whereby direct microscopic examination of the gingival vessels of the living animal could be made with comparative ease; accurate interpretation of the vascular conditions was possible on the basis of the previous histological study on *post mortem* material from other animals.

Recently I have been able to make photographic records of the capillaries *in vivo* and a few of these are shown in Figure 1 (photographs 1, 2 and 4), a brief description of each illustration being given in the appropriate figure legend (King, 1944, 2). The photomicrographs, the first of their kind to show reasonable detail, are all at the same magnification ($\times 17$) and the marked dilatation and change in capillary pattern with the onset of disease are readily seen; the vessels were not injected or otherwise specially treated. Photographs 3, 5, 6, 7 and 8 ($\times 17$) were taken from animals just after they had been killed with chloroform and before autolytic or other changes had set in; their detail is clearer since longer photographic exposures could be given without fear of movement of the subject. The value of this method of examination is shown by the fact that no disease could be detected with the naked eye in any of the animals from which these photographs were made, with the exception of that shown in photograph 7; this animal had slight hyperaemic swelling of the carnassial gingivae. The same examination technique is now being employed for the human gum in an attempt to discover some standard for normality which can be recorded photographically. Clinical assessment of human parodontal health and the response of diseased tissues to different methods of treatment may then be placed on a less empirical basis and it may be possible to determine more easily the possible relationship of nutritional disorders to various lesions of the supporting structures of the teeth.

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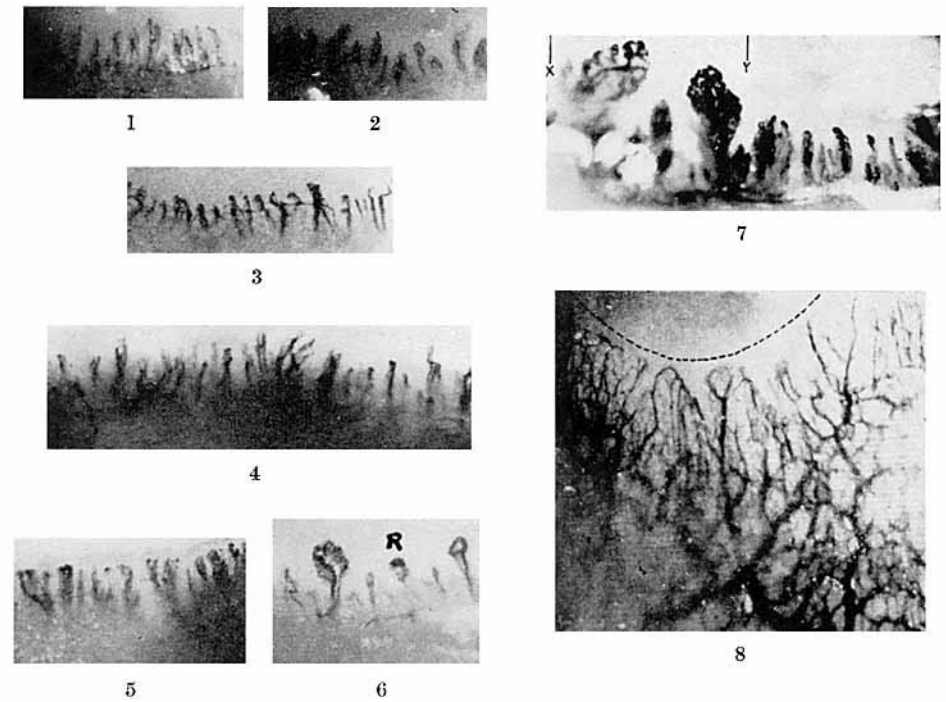

FIGURE 1. SLIT LAMP PHOTOMICROGRAPHS (×17) OF GINGIVAL VESSELS OF FERRETS IN HEALTH AND DISEASE.

1. Normal appearance of carnassial sub-surface gum capillaries *in vivo*. Note regular arrangement of vessels in pairs, in the manner of artery and vein, and small peripheral end bulbs. Each pair of capillaries communicates with its neighbours by fine lateral branches just below the end bulbs and with the larger vessels of the buccal sulcus.

2. Early pathological changes of carnassial region *in vivo*. Note beginning dilatation and loss of characteristic regular arrangement.

3. Early pathological changes of carnassial region 30 minutes after death. Note beginning dilatation and loss of the transverse peripheral communicating branches in some areas.

4. Later stage of disease of carnassial region *in vivo*. Note loss of characteristic capillary pattern, dilatation, and a second layer of capillaries superimposed on the original one.

5. Specimen from animal 30 minutes after death showing stage of disease associated with *microscopic* epithelial hyperplasia. Note resulting bush like appearance of sub-surface capillaries, which may be a defensive mechanism.

6. *Post mortem* specimen of upper carnassial region showing bush formation and "rupture" of vessel at R.

7. *Post mortem* specimen showing varying capillary conditions along the carnassial gum margin. Large bush formations can be seen between X and Y and in this area only could slight swelling of the gum margin be detected macroscopically. To the right of this region, varying phases of capillary reaction are evident although the naked eye appearance of the gum here suggested no disease.

8. *Post mortem* specimen showing normal sub-surface capillaries in the canine region and their junction with the larger vessels of the tissues of the buccal sulcus.

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The Use of Ascorbic Acid in Gingival Repair and in the Healing of Tooth Extraction Wounds

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The classical picture of scurvy from the dental point of view emphasizes sore and bleeding gums, loosening of the teeth, and failure of wounds to heal (Lind, 1772; Price, 1941). This picture, like so many clinical pictures may, however, be modified. Thus Lind states: "The gums were not always affected". The absence of gum changes in infants before the eruption of the teeth and in elderly edentulous patients suffering from scurvy are common clinical observations. In general, however, all observers are agreed that gum changes commonly occur in scurvy. For example Lind writes: "The gums of several bled, being sore and spungy, without their having any other symptom of the scurvy". In experimental scurvy in animals gum changes are commonly seen in the guineapig (Howe, 1920) and in the monkey (Fraser and Topping, 1942). It is worthy of remark that the soft investing tissues of the teeth in scorbutic animals have not received much study.

The Physiological Role of Ascorbic Acid

Before dealing specifically with the effects of ascorbic acid on the dental tissues a short account will be given of the general physiological role, so far as is known at present, of ascorbic acid. This subject is well reviewed by Wolbach and Bessey (1942). They regard ascorbic acid as being necessary for the formation of collagen. Their contention is that: "A failure of the cells to produce an intercellular matrix in scorbutus is the result of the absence of an agent common to all supporting tissues which is responsible for the setting, fibrillation, or jelling of a product which would otherwise remain liquid." They call this the jellation theory of action. Another theory that has been advanced is that failure to form intercellular substances is due to an upsetting of the metabolism of the formative cells (Fish and Harris, 1934). These writers were concerned only with the hard tissues of the tooth. They make no comment on changes occurring in the soft investing structures. In parenthesis it is interesting to remark that they consider that lack of ascorbic acid in the diet may play a role in the development of caries. Two changes observed in scurvy are of some interest as they are somewhat difficult to explain on the basis of defective collagen formation. These changes are hypertrophy of the gums, particularly marked in young children, and bleeding from the gums. The hypertrophy may be regarded as either the result of inflammatory changes in a devitalized tissue leading to a local oedema or, more likely, an actual accumulation of connective tissue cells. This latter process does occur in guineapigs fed on diets inadequate in ascorbic acid for long periods. The process is seen notably at the attachment of muscles to bones and fascia. Wolbach and Bessey (1942) interpret this process as a compensatory hyperplasia. Since no important change in the blood vessels has been described nor have morphological changes been detected in the capillaries in scurvy, the bleeding is

regarded by these same writers as "probably the result of structural weakness, either the result of changes in the cement substance binding the endothelial cells together, or in collagen fibrils immediately adjacent to the capillaries". That ascorbic acid is necessary for the formation of intercellular substance in the guineapig and man is not denied and its importance as an agent in wound healing is now well established (Hunt, 1941; Bourne, 1942).

Why ascorbic acid should be considered of importance for the growth and well being of the dental tissues may be briefly summarized thus: It is needed for the deposition of the intercellular matrices of connective tissue, dentine and bone.

The Use of Ascorbic Acid in "Gingivitis"

The relation of dietary ascorbic acid to the condition known as gingivitis is of general and topical interest. Gingivitis, if it is a distinct clinical entity, means, rather vaguely, inflammation of the gums. The aetiology is manifold as can be seen from Table 1, which makes no claims to completeness. An excellent account of the subject is given by Mead (1940).

TABLE 1

THE AETIOLOGY OF "GINGIVITIS"

Type	Causes
Traumatic	Lack of oral hygiene Calculus Masticatory stresses on probably devitalized tissues Mouth breathing
Infective	Streptococcal infection Infection with "Vincent's organisms"
Nutritional (McCollum, 1941)	Deficiency of vitamin A vitamin C vitamins of the B_2 complex
Metabolic	*Diabetes mellitus*
Toxic	Poisoning by heavy metals, *e.g.*, mercury or bismuth Action of Dilantin (diphenylhydantoinate)
Physiological	Tooth eruption

We were associated in the treatment of certain cases of gingivitis in Dundee, which responded well to the administration of ascorbic acid. The lesion of the gums was pronounced and, be it marked, was the admitting complaint of the patient. We have not, as yet, made an investigation for slight degrees of gingivitis in patients at the Dental Hospital. Our diagnosis of simple gingivitis is that the patient complains of sore and bleeding gums. The gums are inflamed but there is little or no production of pus. On digital pressure the gum bleeds. We do not intend here to discuss ulcerative gingivitis which we consider distinct from simple gingivitis. Further details have been published (Campbell and Cook, 1942, 2). Ascorbic acid was used in treatment as it seemed

the logical remedy. We had read Hanke's (1930) paper and thought the subject worth re-investigation. The dietary level of ascorbic acid of the artisan class in Dundee is generally low. Investigations by Cook, Davidson, Keay and McIntosh (1944) on a group of boys showed an average intake of 19 mg. per day, not allowing for losses in cooking except in soups. The level of plasma ascorbic acid was found to be correspondingly low.

With regard to the efficacy of ascorbic acid in treating cases of "gingivitis" there are workers who have obtained beneficial results, and other workers who regard it as of no value, or again there are those who are undecided. Some of the exponents of these differing views are listed in Table 2. An experiment that is usually quoted to discredit the idea that gum changes indicate ascorbic acid deficiency is that of Crandon, Lund and Dill (1940). Although skin changes of a hyperkeratotic nature

TABLE 2

OPINIONS ON THE VALUE OF ASCORBIC ACID IN THE TREATMENT OF "GINGIVITIS"

For	Against	Undecided
Howe (1920)	Fox, Dangerfield, Gottlich and Jokl (1940)	Fitzsimmons (1941)
Hanke (1930)	Crandon, Lund and Dill (1940)	Burrill (1942)
Westin *et al.* (1937)	McNee and Reid (1942)	Radusch (1942)
Kramer (1937)	Ungley and Horton (1943)	King (1944)
Roff and Glazebrook (1940)	Kohn, Milligan and Wilkinson (1943)	
Blockley and Baenziger (1942)	Macdonald (1943)	
Campbell and Cook (1942, 2)	King (1943)	
Faber (1942)		
Kruse (1942)		
Kent (1943)		
Stuhl (1943)		

were observed when the level of plasma ascorbic acid fell to very low values, Crandon's gums were not affected. The most striking feature of this case was that an experimental wound that had been inflicted failed to heal. On what would have happened to a wound on his gums, it is interesting to speculate. Apparently Crandon's teeth and their supporting tissues were very good initially and had been well tended.

To try to bring harmony among these discordant opinions we suggest that among very many other factors the following should be given serious attention: (1) The manifold aetiology of the condition and (2) the question of individual variation in the need for ascorbic acid. As stated above, gum changes are not seen in all cases of scurvy or, again, in all persons whose level of plasma ascorbic acid is well below 0·2 mg. per 100 ml. As a personal view we re-iterate the statement we made in 1942 for cases seen at the Dundee Dental Hospital: "The primary cause of simple gingivitis is difficult to determine. For our purposes it may be regarded as either (*a*) the manifestation of an ascorbic acid deficiency state, or (*b*) as due to the presence of wounds on the gum caused by some traumatic agency. If the view is taken that gingivitis is primarily due to trauma we may regard the ascorbic acid as acting as an agent in wound healing." (Campbell and Cook, 1942, 2).

Taking the view that many cases of gingivitis are due to trauma, particularly when associated with a sub-scorbutic state, several common sense procedures suggest themselves:

(1) Removal of the cause of trauma. This is an established surgical procedure. The commonest cause of trauma would appear to be the presence of calculus.

(2) Removal of any focus of infection, if present. Ascorbic acid is not an antiseptic, nor has anyone, as yet, claimed anti-biotic properties for it.

(3) Attention to the dietary regime of the patient, particularly ascorbic acid if the intake is low. As stated above, many persons in Dundee show very low intakes of this substance.

The great value in the diet of properly cooked green vegetables and roots should be stressed. Only in severe cases should the ascorbic acid be administered as tablets.

Obviously all dietary factors should be attended to, because the intercellular substance is protein in nature and is produced as the result of the activity of actively functioning cells. These cells need for their proper functioning other vitamins, particularly vitamins A, D and members of the vitamin B_2 complex. The importance also of the inorganic constituents of the diet needs no stressing.

Recurrence Rate. Unfortunately data on the important subject of the recurrence rate after different forms of treatment is scanty. It was impossible to determine our own rate, but the impression we received was that it was low. Macdonald (1943) gives a value of 22 per cent. as a recurrence rate 3 months after local treatment (scaling) only.

The Use of Ascorbic Acid in the Healing of Tooth Extraction Wounds

Considering the dietary intake of ascorbic acid generally found in patients at the Dundee Dental Hospital, and the favourable results we had obtained in treating cases of "gingivitis," the obvious experiment of testing the action of ascorbic acid in assisting in the healing of tooth extraction wounds suggested itself. Preliminary experiments gave very favourable results. To place the matter on a more secure basis the following procedure was adopted. A patient was selected in whom multiple extractions were necessary. The teeth in one half of one jaw were removed without any dietary preparation of the patient. Impressions of the jaw were then taken immediately after extraction and again after an interval of 24 hours. Seven days later the patient was given ascorbic acid orally and the teeth on the other side were removed, an impression being taken, and then again after an interval of 24 hours. A comparison could thus be made of the healing process after 24 hours, without treatment, and after the administration of ascorbic acid. This procedure has been carried out on numerous patients with uniformly successful results (Campbell and Cook, 1942, 1). What struck us most in these cases was the rapid healing of the gum tissues and the rapid absorption of the alveolar process when ascorbic acid was given.

An experimental study of the healing of tooth extraction wounds was carried out by Claflin (1936), who found the following sequence of events.

Blood clot fills the socket. The gingival tissues collapse over the wound and form a protective covering for the clot, which now undergoes a process of organization. By the fifth or sixth day the epithelium has grown from the periphery and completely covers the clot. On the seventh day the first evidence of bone formation is found along the walls of the socket. Bone is laid down in delicate trabeculae which come from the original alveolar bone and proliferate into the organized clot. Simultaneous with bone formation in the socket is a process of bone resorption along the crest of the alveolus. As a result the socket is being filled with new bone at the same time that its depth is being decreased, until the newly formed bone has reached the level of the resorbed crest.

The need for ascorbic acid, *inter alia*, in such processes, where so much intercellular substance is being laid down, needs no stressing. There is some evidence of an additional, possibly pharmacological, action of ascorbic acid and that is that the amount of haemorrhage after extraction is reduced to a minimum.

Summary

Ascorbic acid is of great value in treating certain forms of gingivitis, particularly if the dietary history of the patient suggests that there is a deficiency of this vitamin. The writers regard the ascorbic acid as assisting in the wound healing of gingivae damaged by trauma. It may be that slight traumatic agencies have an effect in some ascorbic acid deficient patients out of all proportion to the trauma involved. The importance of combined local treatment is stressed.

It is suggested, however, that ascorbic acid is only one dietary factor, lack of which will give rise to gingivitis. In treating cases of this condition generally, the dietary as a whole should, as far as possible, be made up to optimal values.

The value of ascorbic acid in the healing of tooth extraction wounds would appear to be undoubted.

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Chronic Fluorine Intoxication in Sheep and its Effect upon the Teeth

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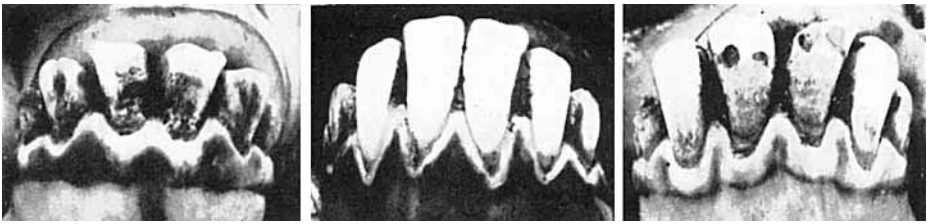
Introduction

A very large number of papers has been published dealing with fluorine poisoning in domestic animals. These were very ably dealt with in an exhaustive monograph by Roholm (1937), and were analysed in a more concise form by Steyn (1938). Fluorine poisoning of domestic animals has most often been of a chronic type, and is readily divisible into two main groups. The first group contains those instances in which the poisoning has occurred under natural conditions. The second group includes those instances that have resulted from the activities of mankind.

In the first group we have those forms of fluorine poisoning that result from the native presence of fluorides. Perhaps the outstanding example of this form is the condition known as "*Darmous*" that occurs in north Africa, in Algeria, Tunisia and Morocco. The aetiology and pathogenesis of the disease in these areas were extensively investigated by Velu (1933), who proved that the water supplies were contaminated in their passage over phosphatic strata containing fluorides. Similar occurrences have been reported also from certain areas in north America and from some other parts of the world. In these cases sheep were the animals principally affected; cattle were less susceptible and horses appeared relatively resistant. Another interesting instance of naturally occurring fluorine poisoning is that which followed the eruption of Hecla in Iceland.

In the second group we have the cases of fluorine poisoning that have occurred as the result of local contamination of herbage from industrial activities. Cases occurring in the neighbourhood of aluminium factories have been reported from Norway, Switzerland and Italy. A particularly clear record of the condition in the neighbourhood of an aluminium factory in north Italy is given by Bardelli and Menzani (1935). In this country, as far as I know, the only recorded instance of the disease occurring as a



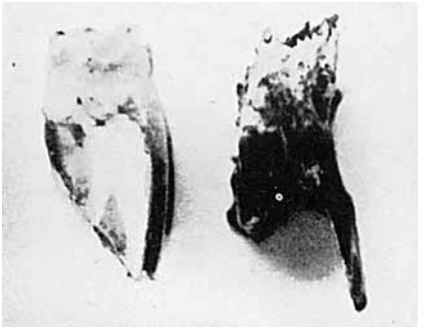
2. Affeeted sheep showing mottling of enamel and deformity of lateral tooth.

1. Normal sheep.

3. Affeeted sheep showing mottling and pitting of enamel.

INCISOR TEETH.

4. Outer aspoet of eheek teeth of affeeted sheep.



5. Isolated molar tooth normal and affeeted.

FIGURE 1. PHOTOGRAPHS OF NORMAL AND AFFECTED TEETH.

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result of contamination of pastures from the manufacture of aluminium is that which I propose to discuss. Fluorine poisoning of stock has been described as occurring in an area in England in the vicinity of brick works (Blakemore, Bosworth and Green, 1941). The possibility exists of fluorine poisoning occurring among animals grazing on pastures that have been heavily top-dressed with north African phosphates containing fluorides; some work has been done on this, but the results are not entirely conclusive.

Clinical Details

The cases of fluorine poisoning that I have investigated occurred in the immediate neighbourhood of an aluminium factory. A croft and two farms were involved. The croft is nearest to the factory, being about half a mile from it. Examination of the stock showed that all the adult sheep that had been reared on the croft were seriously affected, but the lambs were free. On the farm which is about three-quarters of a mile from the factory, 62 per cent. of the sheep examined were affected, and on the farm about half a mile farther from the factory 46 per cent. of the sheep examined were affected.

The dental lesions found in the sheep may be described as taking 3 forms. First, there is mottling of the enamel of the permanent incisor teeth. In sheep there are 4 pairs of permanent incisors, the lateral pair really being incisiform canines; the central pair of permanent incisors erupt at 15 months, the next pair at 21 months, the next at 2 years 3 months, and the lateral pair at 2 years 9 months. In addition to mottling, the incisor teeth may be deformed, or may be so brittle that they readily break, or may have so little resistance as to wear down rapidly to stumps level with the gums (Figure 1, Nos. 1, 2 and 3).

Second, there is the poor wearing quality of the cheek teeth; this leads to selective abrasion and deformity. As the cheek teeth do not meet in perfect apposition, the grinding action of feeding and ruminating causes the teeth to wear on a slant so that long pointed projections form on the outer aspect of the upper teeth and the inner aspect of the lower teeth. The abnormal wear of the cheek teeth is most pronounced in the 1st and 2nd molars. These long points impinge on the gum of the opposite jaw, causing ulceration and permitting the entrance of infection. In many cases the formation and eruption of the teeth have been irregular, leading to deformity of the mouth, and this, with the irregular wear, produces a state of the teeth that renders it impossible for the animal to chew its food properly. A considerable quantity of food may be ingested, but this is not efficiently ground during the process of rumination and much of it is lost from the mouth during the animal's unsuccessful attempts to chew the cud. The process of selective abrasion sometimes exposes the pulp, causing intense pain that prevents the sheep feeding, and also provides another portal of entry for infection (Figure 1, Nos. 4 and 5).

Third, the entrance of infection leads to an alveolar periostitis and suppuration of the roots of the cheek teeth; this through the sinuses of the skull produces a chronic purulent nasal discharge. I have found when dissecting skulls for analysis that there was pronounced suppuration at the roots of all the cheek teeth in a high proportion of affected animals. It is possible to detect badly affected sheep by selecting those with a

purulent nasal discharge. In a proportion of cases, in addition to dental lesions, I have found evidence of osteoporosis; in several cases the ribs were exceedingly fragile and one of the skulls showed marked osteoporosis.

Chemical Analysis

In order to establish that the conditions I have described were due to the effects of chronic fluorine intoxication, a number of analyses were made by my colleague, Professor Thin. The results of these analyses I may summarize as follows: The teeth of affected sheep had a fluorine content ranging from 0·25 to 0·86 per cent., compared with 0·15 to 0·19 per cent. in a series of normal sheep. The bone of the jaws of affected sheep had a fluorine content of from 0·30 to 1·25 per cent., compared with 0·19 to 0·27 per cent. in the series of normal sheep.

Analysis of herbage from the croft showed the remarkably high figure of 61 p.p.m. of fluorine. Herbage from the neighbouring farm had a fluorine content of 44 p.p.m., and the soil from this farm contained 51 p.p.m. Running water from a stream on the croft contained 0·1 p.p.m. of fluorine. Slagsvold (1935) found that hay in the neighbourhood of aluminium factories in Norway contained 50 p.p.m. of fluorine.

Discussion

Though the mottling of the enamel of the incisor teeth is a characteristic feature of chronic fluorine poisoning which is of value in identifying the condition, I do not consider that of itself it has any serious harmful effect on the sheep. Dental caries is extremely rare in sheep, and there is no evidence that its incidence is increased by the action of fluorine. The optimum concentration of fluorine in water to prevent caries is thought to be in the neighbourhood of 1 p.p.m. It has been stated that in the human subject, if the fluorine concentration is above 4 p.p.m., there is a danger of a severe degree of mottling with pitting. In these cases in sheep the fluorine concentration in the food was very high. It is well to point out that mottling of the incisor enamel was not constantly present; I have encountered cases with marked evidence of damage to the cheek teeth which showed no mottling of the incisor enamel, but were proved by *post mortem* examination and analyses to be cases of fluorine poisoning. The poor wearing quality or deformity of the incisors may interfere with the sheep grazing; this has a more serious effect when the sheep is grazing on relatively short pasture. The serious effects resulting from the selective abrasion of the cheek teeth can readily be appreciated. In many cases the sheep becomes extremely debilitated and may be quite unable to withstand a spell of severe weather. Lambs may be born weakly and the ewe has little or no milk for her offspring. A very striking feature of these cases of chronic fluorine intoxication has been the complete absence of either clinical or *post mortem* evidence of any contributory cause of the debility. The very low helminth burden was of particular note.

I have pointed out that it is most often the first or second molar teeth that are seriously affected by the process of selective abrasion. The first permanent molars in the sheep erupt at between 3 and 5 months of age, and the second molars at between 9 and 12 months; the third molar and the premolars erupt at between 18 months and 2 years. The

deciduous teeth are not affected as they have erupted before the lamb starts to graze, and fluorine does not pass from the maternal circulation to either the foetus or the milk. The effect of the suppurative process I have described can readily be appreciated.

The relationship of the fluorine content of the herbage to the dental changes is of interest. It has been stated by Dean (1936) that 10 per cent. of children drinking water containing 1 p.p.m. of fluorine show mottling of the enamel, and 100 per cent. of children show this change in their permanent teeth if the water contains 10 p.p.m. On the croft 100 per cent. of the sheep were affected, and their sole foodstuff contained 61 p.p.m. of fluorine.

Finally, there is the question of remedial measures. There is no known antidote to fluorine. If the sheep farm involved could be run on a system whereby all the lambs were sold off at weaning time and adult ewes were bought in to maintain the stock, the condition would be avoided. This method could be applied to a small enclosed croft or grazing, but it could not be used on a large sheep farm with an open hill, as adult ewes would not settle to the grazing. The proper remedy is, without doubt, for modifications to be made at the factory to mitigate the nuisance. This is an engineering problem beyond my sphere, but I am told that it is neither impossible nor impracticable.

ACKNOWLEDGEMENTS

I am indebted to my colleague Professor R. G. Thin for the analysis quoted in this paper and to Mr. K. A. Walker, M.R.C.V.S., for the photographs used to illustrate it.

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Diet and the Calcified Dental Tissues

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The calcified dental tissues are enamel, dentine and cementum. Enamel is the hard, glistening tissue covering the crowns of the teeth in man and most mammals. It is the hardest animal substance and contains less organic matter than any other tissue of the body. It is composed of enamel rods or prisms and a calcified substance, which unites the prisms into a continuous structure, called the interprismatic substance. Enamel contains approximately 90 per cent. of calcium phosphate with traces of fluoride, the remainder being composed of smaller amounts of calcium carbonate, magnesium phosphate, other salts, cartilage and fat. Dentine is a connective tissue whose intercellular substance is calcified. It is penetrated by minute tubules which contain protoplasmic fibres

from cells lying within the pulp chamber and from the pulp itself. Dentine contains 66 per cent. calcium phosphate with traces of fluoride and 27 per cent. organic matter; the remainder is as in enamel. Cementum is also a connective tissue whose intercellular substance is calcified. It is arranged in layers around the tooth root. Its function is to attach to the tooth the connective tissue fibres or peri-odontal membrane from the surrounding alveolar bone.

The effect of diet on the calcified dental tissues may be either local or systemic. It must be considered in relation to the 3 main periods of dental life; the ante-natal period, including the effect on mother and foetus; the post-natal period until the growth of teeth is complete, and the last period when teeth are fully formed and erupted.

The two main diseases or defects to which the calcified dental tissues are prone are hypoplasia or defective structure and dental caries or decay.

Hypoplasia

Hypoplasia is a condition which arises during the ante-natal and post-natal periods but before eruption of the teeth. It is an upset of calcification and is characterized by poor enamel structure and the formation of interglobular spaces in dentine.

At one time this term was used to denote only obvious gross abnormalities in tooth structure, but the work of Mellanby and King (1939) has shown that the term should be used to denote any observed deviation, however slight, from perfect structure. Their clinical basis for detection of hypoplasia is by probe examination. The naked eye appearance of teeth may not seem to differ appreciably, but if the teeth are dried and illuminated by oblique light, variations in surface structure of the enamel are strikingly defined. Very few teeth are found to have perfectly smooth, shiny surfaces of good colour. Defects vary from slight irregularities to minute pits and grooves, and up to gross defects.

Many writers have shown that gross hypoplasia, which is relatively infrequent, bears very little relationship to caries, but Mellanby (1934; Committee for the Investigation of Dental Disease, 1936) has demonstrated that the much commoner defects, detectable by the probe as minor irregularities of the enamel surface, are important in causing predisposition to caries. This condition has been called M- (or Mellanby) type hypoplasia, as against the less common G- (or gross) type. How then does diet enter into this picture of hypoplasia? Remembering that hypoplasia is a state of the ante- and post-natal periods but before eruption, it will be seen that diet can influence the condition only through systemic effects.

Animal Investigations

Most of Mellanby's early experiments were made on puppies. It was observed that diets lacking egg yolk, milk, animal fat and cod liver oil gave rise to teeth defective in structure. Such defects were apparent in the tissues formed during the period of experimental feeding and were characterized by rough, pigmented, hypoplastic enamel, faulty dentine and poorly calcified bone. When a liberal supply of these foods was added to the same basal diet, abnormalities were absent. Attention was then paid to the effect of the maternal diet on the teeth of the offspring.

When this diet was rich in fat soluble vitamins, the deciduous teeth of the puppies were well formed, while vitamin deficiency resulted in deciduous teeth of poor structure (Mellanby, 1918, 1922–23, 1927, 1929, 1930). It has to be pointed out that in dogs all the deciduous teeth are usually erupted and the crowns largely calcified at the time of weaning at 5 to 6 weeks. Therefore their structure depends mainly on the supply of the necessary material during pregnancy through the placental blood supply, and during lactation through the milk. A sufficiency of calcium and phosphorus is of course essential.

Investigations on Man

Toverud and Toverud (1931) from their metabolism experiments in a home for expectant mothers concluded that a diet deficient in calcium, phosphorus and vitamin D is an important predisposing cause of hypoplasia and caries in children. Mellanby's (1934) studies of children's teeth showed that varying degrees of M-hypoplasia were found in 60 per cent. of deciduous teeth obtained from private sources and in 87 per cent. of teeth obtained from children attending elementary schools, indicating that the majority of children's deciduous teeth are defective in structure. Furthermore, the teeth of poorer children appeared more defective in this examination than those of children in better circumstances. Later, Mellanby (Committee for the Investigation of Dental Disease, 1936), working with a greater number of children, confirmed the earlier findings. Further evidence has been presented by King (1940) in his work on the Island of Lewis, on the relationship between diet and M-hypoplasia. In surface structure the teeth of Lewis rural children were found to be much superior to those of the urban children. This was especially the case in the coastal districts where more fish was eaten. Cathcart, Murray and Beveridge's (1940) quantitative study of the diet of Lewis families has demonstrated that the diet of the rural children included a relatively high proportion of fat soluble vitamins and mineral salts. It is considered that this diet provided a more abundant supply of the nutritional factors necessary for the proper development and calcification of the teeth.

M-hypoplasia then is a structural defect of enamel and dentine, the governing factor in its initiation being mainly dietary deficiency in the ante- and early post-natal periods. Measures for its control must first be directed towards a well balanced maternal diet, rich in fat soluble vitamins A and D and with an adequate calcium and phosphorus content. The content of ascorbic acid, which affects the formation of the intercellular elements and odontoblast layer of the dental pulp, must also be considered. Finally this attention to diet must be continued throughout the post-natal period until formation and growth have been completed. Hypoplasia and delayed eruption of teeth occur in rickets. In rachitic teeth the crowns are stunted, the hypoplasia is of the "gross" type and the disease mainly affects the permanent dentition. The widespread use of vitamin D in the prophylaxis of rickets is now such that the dental signs of the disease are seldom seen.

Dental Caries

Dental caries is a disease of post-natal life and occurs after the teeth have erupted. It is a disintegration of tooth tissue extending

progressively from without inwards towards the pulp. Histological examination shows this to be the case. Enamel is attacked first, then dentine and ultimately the pulp.

If diet is to be considered as a factor in the initiation or inhibition of caries, does it act locally or through the general system?

Systemic Effects

Enamel and dentine, unlike bone, are not reserve sources of calcium. Bone is normally subject to calcium withdrawal to supply other needs in the body, but teeth have no vascular or cellular mechanisms like those found in the tissues round bone for osteoclastic removal of calcium. The average person has from 1400 to 2000 g. of calcium in the body, 12 g. of this being in the permanent dentition. The body of the newborn infant contains about 24 g. of calcium, *i.e.*, 1 or 2 per cent. of the skeletal supply of the mother. If the calcium intake during pregnancy is adequate for general health, there will be no loss from the mother's bones, and even in severe deficiency, the loss of tooth calcium seems hardly possible. Pregnancy is not a direct cause of caries in the mother. Schour and Massler (1943) have published statistical evidence to show that no significant difference is found in the incidence of caries between pregnant and non-pregnant women of corresponding age. If the cause were systemic it is reasonable to ask why caries does not occur before eruption of teeth. Any disturbance in calcium metabolism during tooth development, either intra- or extra-uterine, may result in dental hypoplasia in the infant or child, but not in caries.

The Committee for the Investigation of Dental Disease (1936) states that a "relatively high vitamin D content of the food can do much to diminish the incidence of caries if the vitamin is given during the development of the teeth" (undoubtedly so by its effect on tooth structure); that "a beneficial effect may be obtained if the vitamin is given at a fairly late stage of development" (again through tooth structure); and that "even when it is given after the eruption of the teeth, the onset and spread of caries is delayed". My comments are in brackets; with the last statement I cannot agree. It has not been proved that vitamin D can inhibit the spread of caries after complete growth and eruption of the teeth.

Local Effects

Dental enamel is the most highly mineralized tissue in the body. It is generally held that there is only one means, apart from trauma, by which enamel can be destroyed and that is by the action of acid. *In vitro* experiments have shown that enamel can withstand immersion in an acid solution with a *p*H value of 5 to 5·5, but that at a *p*H below 5 decalcification takes place. If a section of enamel is treated with 2 per cent. hydrochloric acid for a few seconds, mounted and examined, it will be seen that the prisms stand out very distinctly. This is due to the dissolving of the interprismatic substance and the picture in some ways simulates the first signs of enamel caries.

Is it possible that the *p*H of saliva may fall so low as to become dangerous? In a study (Whyte, 1941) further described below, in an institution, I estimated by a colorimetric method the salivary hydrogen

ion concentration of 50 boys. The tests were made between 12 and 12.30 p.m., *i.e.*, before the midday meal and on resting saliva, so that the effect of food should be reduced to a minimum. The lowest *p*H found was 6·5, the highest 7·4. The average for the 50 boys was 6·96. Admittedly there is an experimental error in colorimetric methods of determination, but there is a distinct gap between the figure of 6·9 and the critical decalcifying *p*H level of enamel of approximately 5·0. How then is the *p*H of 5·0 or lower reached in the mouth and does diet influence it? Of the 3 chief constituents of food, protein does not undergo digestion in the mouth and fat does not appear to undergo any change. Hence, if one of these constituents is concerned, it must be carbohydrate.

Miller (1890) proved that there were present in the human mouth micro-organisms which, when cultivated in suitable media, were capable of producing lactic and other acids. Test tube mixtures of saliva and bread, or other carbohydrate food, fermented on incubation at body temperature with the production of acid. If extracted teeth were placed in such mixtures the enamel became decalcified and assumed an appearance somewhat similar to that seen in early caries. It was inferred that a similar fermentation and enamel decalcification occurred in the mouth. Miller's theory may be summarized thus: caries is a process of dissolution of the calcium salts from enamel and dentine by acids formed in the mouth by fermentation by bacteria of carbohydrates lodging on tooth surfaces. This process is followed by disintegration of the organic matrix by proteolytic action of the same or other bacteria. Bunting and Palmerlee (1925) in America claimed that susceptibility to or immunity from caries can be forecast from saliva by the presence or absence of members of the *B. acidophilus* group of micro-organisms. The more recent publications of Bunting (1936, 1939), however, have pointed to a correlation between *B. acidophilus*, increased sugar in the diet and caries production. It is still maintained that *B. acidophilus* is the causal organism, but it is also stated that the incidence of the disease may be greatly modified by reduction of the carbohydrate intake. Unfortunately Bunting has produced no system for the assessing of caries data to support his contentions.

Some 6 years ago I undertook a dietetic, dental and bacteriological study of 50 inmates in a boys' institution (Whyte, 1941). The ultimate aims of this investigation were twofold: (1) To note if any correlation could be found between an increased carbohydrate intake and dental caries development, using a controlled method of carbohydrate administration and a known system of caries assessment, and (2) to note if any association could be found between caries activity and the numerical presence in saliva of the group of micro-organisms generically and, I think, erroneously called "*B. acidophilus*". I could find no correlation whatever between the numerical presence of *B. acidophilus* colonies and caries activity and this part of the investigation proved completely fruitless. Nevertheless I feel that the dietetic findings were significant.

The inmates in this particular institution were not mental defectives. Schooling was given within the institution and no boy was allowed out of the grounds except on one half day a week. There was a standard basal diet, each boy partaking of the same food. Parcels given to the children were examined by the Superintendent and sweets confiscated.

The nearest village shop was 4 miles distant and was considered out of bounds. No outside influence could therefore have interfered with the dietary.

Fifty boys, picked at random, were divided into 2 groups of 25, each group in turn acting as control. Additional carbohydrates were given to Group 1 for 2 months, sweets being withheld from Group 2 during this time. At the end of this period the positions were reversed, Group 2 receiving sweets for 2 months. This complete cycle was repeated again so that both Group 1 and Group 2 received an additional 2 months' supply at alternate periods of time.

The sweets chosen were those commonly known as "fudge tablet" and "macaroon bars"; 2 oz. per day was decided upon as being a quantity not greater than that usually consumed. The 2 types were given alternately to prevent the inmates becoming tired of one type. Chemical analysis of both types of sweets was as follows. Fudge tablet: 93 per cent. carbohydrate, 52 per cent. being polysaccharide, 24 per cent. disaccharide and 17 per cent. monosaccharide; macaroon: 85 per cent. carbohydrate, 43 per cent. being polysaccharide, 34 per cent. disaccharide and 8 per cent. monosaccharide. In both instances the polysaccharide was found to be starch, the disaccharide saccharose, and the monosaccharide glucose.

One Medical Research Council type chart was used for each boy, and, in all, 5 clinical inspections were made: the first prior to giving sweets, the second prior to withholding, the third before administering for the second two months' period, the fourth prior to withholding, and the fifth at the end of that two months' period. At the initial inspection all fully erupted teeth were examined and included in the charts. No tooth erupting subsequently was considered. Each cavity present at the initial inspection was given a value according to the severity and extent of the disease process, thus: Discoloured pits, fissures or other areas suspected of being carious owing to the catching of a probe, 1 point; simple cavity larger than grade 1 but not considered to involve more than one-third of the crown, 2 points; cavity thought to involve one-third or more of the crown, 3 points. On each chart, and opposite each carious cavity, the requisite figure was inscribed, if present at the date of initial inspection, in red ink, at the second inspection, green, the third, brown, the fourth, blue, and the last, yellow. By the summation of the points ascribed to each cavity present in any one mouth, a total score for that mouth was calculated. To this the term "total caries figure" (TCF) was given. From this the "average caries figure" (ACF) was obtained by dividing the TCF by the total number of teeth examined. From the individual values, the averages for Groups 1 and 2, and the increases, if any, for each of the 5 periods were calculated. The two tables quoted below show the type of results obtained.

It is apparent that a greater increase occurred in the ACF during periods of administering carbohydrates than when extra carbohydrate was withheld. In Table 2 the figures are rearranged to show the differences between the groups during 2 single periods of 2 months when each was acting as control to the other.

The increase in Group 1 after the second period of administration is appreciably higher than that for the first period of withholding in Group 2. Conversely, the increase in ACF for the second period of administration

to Group 2 is considerably greater than that for the second period of withholding from Group 1. It is believed that these facts are significant.

If carbohydrate fermentation is to be considered an exciting factor in caries initiation, how does it act? The role of *B. acidophilus* as a causal organism has been questioned by many. It has been argued that the members of the *acidophilus* group are opportunists, that they are present in caries active mouths because conditions are favourable for their existence, and that their presence is a result of the carious process rather

TABLE 1

CHANGES IN AVERAGE CARIES FIGURE (ACF) FOR RESULTS OF EXAMINATION OF TEETH OF BOYS RECEIVING AND NOT RECEIVING EXTRA SWEETS FOR ALTERNATING PERIODS OF 2 MONTHS

Time of observation	Group 1		Group 2	
	Total ACF	Increase	Total ACF	Increase
1st Inspection ..	0·561	—	0·371	—
1st Administration	0·801	0·240	0·581	0·210
1st Withholding ..	0·901	0·100	0·612	0·031
2nd Administration	1·123	0·222	0·797	0·185
2nd Withholding ..	1·180	0·057	0·852	0·055
	ACF for deciduous and permanent incisor canine group		ACF for deciduous and permanent incisor canine group	
1st Inspection ..	0·168	—	0·048	—
1st Administration	0·176	0·008	0·074	0·026
1st Withholding ..	0·189	0·013	0·074	0·000
2nd Administration	0·224	0·035	0·119	0·045
2nd Withholding ..	0·251	0·027	0·119	0·000
	ACF for deciduous and permanent pre-molar molar group		ACF for deciduous and permanent pre-molar molar group	
1st Inspection ..	1·043	—	0·696	—
1st Administration	1·448	0·405	1·090	0·394
1st Withholding ..	1·628	0·180	1·134	0·044
2nd Administration	2·042	0·414	1·476	0·342
2nd Withholding ..	2·125	0·083	1·587	0·111

than the cause. In my opinion the members of the aciduric streptococcal group are more likely than *B. acidophilus* to be the initiators of the degradation. Bibby, Volker and Kesteren (1942) state that comparison of the rates of acid formation by various mouth organisms shows that *Streptococci* and *Actinomyces*-like organisms form acids most rapidly, and that *acidophilus* and other organisms are less active. Some streptococcal strains form acid more rapidly than others. Clinically it is not of much importance which organism is indicted since fermentation does seem to take place.

With reference to the kind of carbohydrate involved the work of Osborn, Noriskin and Staz (1937) is important. They found that South African Bantus, living under native conditions, chewed a great deal of

crude sugar cane, yet were relatively free from caries. As soon as they found work in towns, and adopted a European type of diet, including refined sugar, their susceptibility to caries increased. Experimentally, the authors found that teeth decalcified more readily when immersed in a

TABLE 2

CHANGES IN AVERAGE CARIES FIGURE (ACF) FOUND IN EXAMINATION OF TEETH OF BOYS RECEIVING AND NOT RECEIVING EXTRA SWEETS FOR 2 COMPARABLE PERIODS OF 2 MONTHS

Period	Group 1		
	Treatment	Total ACF	Increase
8.11.39 to 8.1.40 ..	2nd Administration ..	1·123	0·222
9.1.40 to 10.3.40 ..	2nd Withholding ..	1·180	0·057
		ACF for deciduous and permanent incisor canine group	
8.11.39 to 8.1.40 ..	2nd Administration ..	0·224	0·035
9.1.40 to 10.3.40 ..	2nd Withholding ..	0·251	0·027
		ACF for deciduous and permanent premolar molar group	
8.11.39 to 8.1.40 ..	2nd Administration ..	2·042	0·414
9.1.40 to 10.3.40 ..	2nd Withholding ..	2·125	0·083

Period	Group 2		
	Treatment	Total ACF	Increase
8.11.39 to 8.1.40 ..	1st Withholding ..	0·612	0·031
9.1.40 to 10.3.40 ..	2nd Administration ..	0·797	0·185
		ACF for deciduous and permanent incisor canine group	
8.11.39 to 8.1.40 ..	1st Withholding ..	0·074	0·000
9.1.40 to 10.3.40 ..	2nd Administration ..	0·119	0·045
		ACF for deciduous and permanent premolar molar group	
8.11.39 to 8.1.40	1st Withholding ..	1·134	0·044
9.1.40 to 10.3.40 ..	2nd Administration ..	1·476	0·342

mixture of saliva and refined sugar than when crude sugar juice was substituted. From this they postulated that the crude substances effected some protection against decalcification. The protective substances were found to be the minerals, calcium and phosphorus. They further proved that the protective action of crude sugar could be imitated *in vitro* by the addition of 1 per cent. calcium glycerophosphate to the

refined sugar. Murray (1939) working in the same field in this country showed that, while "white sugar" contained neither mineral, crude and less refined varieties contained varying amounts of both calcium and phosphorus. Sugar in solution cannot lodge on the teeth but starches are comparatively insoluble in the mouth and can lodge round teeth and form a plaque with bacteria and mucin. Sugar can then be used by the colony to increase acid production, with a more rapid and destructive effect.

How can this acid production be determined? Here I think sufficient weight has not been given to the work of Stephan (1940) and Stephan and Miller (1943) in America. They state that "regardless of whether the nature of the caries producing plaque is bacterial, gelatinous, mucinous, composed of food particles or any combination of these, theoretically it is necessary for the plaque to develop a sufficient hydrogen ion concentration to dissolve enamel, in order for the carious lesion to develop". They have been able to measure the *p*H of plaques *in vivo* by electrometric methods, using an antimony electrode. A potentiometer electrometer was used to measure the potential of the system and readings were made directly in *p*H units. Before using in the mouth it was standardized with a buffer of *p*H 4·6. The results are startling and two examples will serve to show their nature.

(1) At 10.55 a.m. the *p*H of a particular tooth plaque was taken and was 6·8. At 11 a.m. a 10 per cent. saccharose mouth rinse was given for 1 minute. The *p*H was recorded at 11·4 a.m. as 4·5; 45 minutes later the *p*H had risen to the original value of 6·8. With a 10 per cent. glucose rinse and at the same periods of time the *p*H dropped from 6·9 to 4·7. Determinations were made at half-hourly intervals, and in this instance it took 2 hours for the *p*H to return to the original value of 6·9. The same experiments with a lactose rinse did not cause the same reduction of *p*H.

(2) Five carious cavities, opened by operative procedure with rubber dam in place, gave *p*H values between 4·1 and 4·6 without previous treatment with carbohydrate. The most interesting observation was that, within 3 minutes after rinsing, the *p*H may drop over 2 units which represents an increase of hydrogen ion concentration of over 100 times. The indication that lactose solutions do not have this marked effect on plaques suggests that the reaction is specific for certain carbohydrates.

Stephan's further statement that, after thorough brushing of the teeth, the characteristic drop in *p*H does not follow the application of glucose lends considerable weight to the importance of oral hygiene as a prophylactic measure. I feel that, once additional research from independent sources has corroborated Stephan's work, this may prove to be the missing link in the chain of evidence.

Finally, I should like to draw attention to data obtained by Wheatley (1921). He showed the difference in the incidence of caries in Shropshire before 1914 and in 1919. The percentage of children free from caries at the age of 5, before 1914, was 5·0; in 1919 it was 44·0. At the age of twelve, in 1914, the figure was 2·9; in 1919 it was 27·1. His methods of assessing caries are probably different from those of today, but the figures are sufficiently illuminating. Wheatley concludes by saying that the lesser incidence of caries in 1919 was due to the "restriction and modification of food during the war, sugar, bread and milk, the wholesale

cutting down of sugar and the almost total elimination of sweets was the most powerful factor." A statistical survey at the end of this war might bring forth some comparable results.

Summary

Attention to diet and its vitamin content in the ante-natal and post-natal periods, until the formation of teeth is complete, can do much to lessen the incidence of dental caries, through the effect of diet on tooth structure. After the period of complete growth has been reached, nutritional factors can play only a secondary role and oral hygiene is in the main line of defence.

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Discussion

Dr. A. J. Glazebrook (Royal Infirmary, Edinburgh): Data from an experimental study of plasma ascorbic acid in boys in a Glasgow reformatory showed some correlation with the incidence of gingivitis.

Dr. I. Leitch (Imperial Bureau of Animal Nutrition, Bucksburn, Aberdeen): Will Dr. King describe the diet of the ferrets he used. What was the difference in diet between the ferrets with healthy teeth and the ferrets with imperfect teeth?

Dr. J. D. King: The animals were from a stock colony and were given every food factor thought to be necessary. Animals picked out from the stock colony showed different phases or stages of the lesions. The actual aetiology is unknown. More work is required. The diet was of meat, milk, cod liver oil and yeast.

Speaker (not known): What effect has ascorbic acid on gingivitis when employed after pockets in the gums have formed? Real recovery occurs when the presence of the disease is appreciated in the early stages.

Professor H. G. Campbell: It is not claimed that ascorbic acid cures. It does not necessarily replace local treatment.

Professor L. S. P. Davidson (Department of Medicine, University New Buildings, Edinburgh): I have seen a case in which there were septic teeth and frank scurvy but no bleeding gums.

Dr. H. Scarborough (Royal Infirmary, Edinburgh): It is not claimed that low blood ascorbic acid in itself causes gingivitis. Although there is no clear correlation between gingivitis and low plasma ascorbic acid it is possible that these two are related through a third unknown factor. Attempts are being made to identify some other factor, possibly nutritional, operating to produce this correlation.

Dr. E. C. Owen (Hannah Dairy Research Institute, Kirkhill, Ayr): Can Professor Boddie state whether the growth of teeth was inhibited by fluorine and whether the analysis of the pasture had been made before or after washing. As far as I know there is no antidote to fluorine poisoning.

Professor G. F. Boddie: The growth of the teeth was definitely checked, as shown by X-ray, but in the vast majority of cases the teeth were completely formed. What would be the use of washing the pasture before analysis? Sheep do not wash their food. Human beings do not get fluorine poisoning from vegetables because they are washed and the water thrown away and with it the fluorine. What is happening is the constant contamination of the pasture by the fumes. In the case of hay you have contamination of the grass during growth, contamination of the hay during hay making and contamination of the hay in the rick.

Dr. E. C. Owen: I am afraid the question has been misunderstood. In analysing plant materials one very often wants to know whether the substance is a protoplasmic constituent of the material examined or whether it is due to soil contamination. I think it would be more usual to wash things than not to.

Dr. G. Dunlop (Auchincruive, Ayr): Pigs and horses are believed to be non-susceptible. Did Professor Boddie find any cases of fluorosis in pigs or horses?

Professor G. F. Boddie: The eruption of the horse's teeth is not significantly different in time from that of the sheep, the sheep's teeth erupting before those of cattle. The ratio of sheep to horses in the district was 2000 to 2. Horses were not bred in the district but imported when fairly old and were not grazing throughout the year. There were no pigs in the district.

Professor H. Dryerre (Royal (Dick) Veterinary College, Edinburgh): I visited the area in question and took blood samples from 6 healthy sheep and from 6 infected animals. The numbers were too small for final conclusions. Five constituents in the samples were estimated, calcium, magnesium, glucose, phosphates and haemoglobin. The figures for

calcium were lower in the affected group. Peirce (1938), in his experiments on the administration of fluorine to sheep, found high concentrations of fluorine in the bones, and teeth were affected most if they had erupted after the animals had commenced to get fluorine. This bears out Professor Boddie's observations that fully erupted teeth are less affected than those which grow during the time of the experiment. The experiments of Peirce also showed that fluorine poisoning had no bad effect on the wool. His figures for the fluorine in soil were much higher than those of Professor Boddie, 700 p.p.m. near to the factory and 20 p.p.m. at a point 4 miles remote. The herbage was not examined.

REFERENCE

Peirce, A. W. (1938). *Bull. Coun. sci. industr. Res. Aust.* no. 121.

Professor G. F. Boddie: Few of the sheep grazed on land very close to the factory. Concerning Professor Dryerre's results for blood calcium it is striking that in chronic fluorine poisoning variable results are obtained according to the amount of fluorine and period of time over which fluorine has been ingested by the sheep.

Dr. J. D. King: There are species differences in tooth development. In rats calcification of the teeth is linked up more closely with the calcium phosphate than with the vitamin D content of the diet. There is a difference between the rat and the pig in their capacity to digest cereals. If the calcium phosphate content of the diet is low and vitamin D is added you get a good effect in the pig but not in the rat.

Professor G. F. Boddie: The picture Dr. Cook has produced in rats given excess fluorine, and shown in his demonstration, differs from that described by other workers who gave yeast in the diet and found that the incisor teeth wore away excessively and one did not get the long "prongs" described.

Dr. I. Leitch: Did Professor Boddie make observations on the skeletons of the sheep and did he find deposits of unorganized bone substance as described by Roholm (1936) in cryolite factory workers.

REFERENCE

Roholm, K. (1936). *Hospitalstidende*, **79**, 981.

Professor G. F. Boddie: The sheep were not lame. Some of the bones had a chalky appearance much the same as those described by Roholm in Iceland sheep. In my own experiments the sheep were getting much less fluorine than those in Iceland.

Several speakers spoke of the deterioration which had taken place in dental health, *e.g.*, among the inhabitants of Tristan da Cunha and of Greenland on the introduction of refined foodstuffs. There was no agreement on the real causes of the dental deterioration, some even blaming the introduction of tooth brushes.

Dr. R. Whyte: With reference to the survey made by Wheatley of caries during 1914 and 1919, the incidence of caries in Shropshire in 1914 was 30 per cent. and in 1919 10 per cent. The fall was attributed to deprivation of sugar during the war years. So far there are no comparable figures for this war.

Dr. J. D. King: Is it not the case that at the present time some people take their whole sweet ration and many are possibly consuming more than they did in pre-war days. In an experiment with 2 children, I gave each of them one sweet at night after brushing their teeth for one year and in the second year one chocolate biscuit. Before the experiment they had 19 carious teeth. At the end of two years the spread of caries was normal and the original caries had been arrested. The effect of sugar on the teeth was open to question.

Dr. I. Leitch: Has any of the speakers made observations on the same lines as Schiøtz (1937) in Oslo, who searched among schoolchildren for those with perfect teeth, starting from the other end of the question. Schiøtz found that it did not matter whether they took or did not take cod liver oil, whether they ate or did not eat sweets or whether they did or did not brush their teeth. The one factor they had in common without exception was that they all ate vegetables and drank milk summer and winter.

REFERENCE

Schiøtz, C. (1937). *Hospitalstidende*, **80**, 57.

Dr. R. Whyte: Could not the incidence of perfect teeth be related to the maternal diet?

Dr. J. D. King: Since 1921 the Medical Research Council has been issuing questionnaires in order to get figures for people with perfect teeth but the answers obtained are unreliable.

Professor W. J. Tulloch (University of St. Andrews Medical School, Dundee): With reference to Dr. Whyte's statements on the Stephan electrometric method of estimating hydrogen ion concentration, the amount of hydrogen ion elaborated in 4 minutes is almost unthinkable from the point of view of fermentation reaction.

Dr. R. Whyte: I checked the figures.

Professor W. J. Tulloch: To me it seems more like an explosion than a simple fermentation.